Research in Psychology and Behavioral Sciences, 2016, Vol. 4, No. 1, 1-6
Available online at http://pubs.sciepub.com/rpbs/4/1/1

DOI:10.12691/rpbs-4-1-1

The Effect of Predisposing Risk Factors of an Eating Disorder on Response Inhibition and Working Memory: An Event-Related Potentials Study

Alison Osborne, Leigh M. Riby*

Department of Psychology, Northumbria University, Newcastle Upon Tyne, UK
*Corresponding author: leigh.riby@northumbria.ac.uk

Abstract A novel investigation was undertaken to assess the effect of a predisposition to an eating disorder on P3a and P3b event-related potentials. Previous research has suggested the P3a and the P3b are reliable markers of inhibitory control and working memory updating, respectively. An opportunity sample of 12 female participants was obtained with mean age of 22.42 (SD = 2.61). Participants completed the Eating Disorder Inventory – 3 assessing their predisposition to an eating disorder along with scores on the included subscales. Response inhibition and working memory was measured using the 3 Stimulus Oddball Task. This task elicits a P3a component in response to novel infrequently presented stimuli and the P3b component in the response to expected infrequent stimuli. Findings showed no evidence of an interaction between an overall predisposition to an eating disorder and P3a and P3b activations. However, results ascertained a significant positive correlation between Body Dissatisfaction scores and the P3a amplitudes. Individuals with a high score on body dissatisfaction scale showed greater activation towards the frontal region than those with a low score during the executive component task, i.e. the greater the score the greater hyperactivity in the frontal area of the brain during response inhibition. With regards to the working memory component, no significant effects were found. Although head maps for body dissatisfaction scores and working memory illustrated that there was a wider spread of activation for the high body dissatisfaction group, rather than concentrated activations in the parietal region.The implications of such results in respect to compensatory activations, the inability to ignore and possible dopamine involvement are discussed.

Keywords: *eating disorder, body dissatisfaction, EEG, ERP,P3a, P3b,oddball task, P300, dopamine, inhibition, executive function*

Cite This Article: Alison Osborne, and Leigh M. Riby, "The Effect of Predisposing Risk Factors of an Eating Disorder on Response Inhibition and Working Memory: An Event-Related Potentials Study." *Research in Psychology and Behavioral Sciences*, vol. 4, no. 1 (2016): 1-6. doi: 10.12691/rpbs-4-1-1.

1. Introduction

Eating disorders are frequently seen as a new age phenomenon when in fact they have been evident throughout history in various guises. One of the earliest indications of eating disorders was in the 13th century with self-starvation among religious groups [1]. Eating disorders are common and relatively chronic conditions, primarily affecting young women [2]. They are characterised by disordered eating behaviours where the individuals' attitude towards weight and shape as well as their perception of body shape are disturbed [3]. Individuals with eating disorders have difficulty tolerating negative experiences and distress, and use food, whether in an over-restrictive or over-consumptive manner to block out awareness of emotions that cannot be tolerated by the individual [4]. The three main eating disorders are defined; Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge Eating Disorder (BED) [3].

The present consensus defines eating disorders as multi-faceted, multi-determined, psychiatric disorders resulting from a complex interplay between biological, psychological and socio-cultural factors. It is this that makes it difficult to determine the cause. Research suggests many factors that contribute to the onset of an eating disorder, such as the role of social pressures to be thin and body preoccupation [5], chronic low self-esteem [6], elevated dietary restraint [7,8] and maladaptive perfectionism [9]. Many predisposing factors are evident across all eating disorder types. The importance to identify individuals at risk is to help aid in preventative measures against the development of eating disorders; across disorder types.

According to the dual-pathway model of eating pathology [10] perceived pressure to be thin and the internalisation of such ideals produces body dissatisfaction. Body dissatisfaction theoretically promotes unhealthy dieting behaviours such as restriction that may progress to AN. Periods of dietary restriction may appear to permit binge eating without gaining weight, which may endorse a cycle of acute restriction punctuated by overeating, characteristic of BN. Dieting might also promote binge eating because violating strict dietary rules can result in disinhibited eating (the abstinence-violation effect).

Moreover, dieting entails a shift from reliance on physiological to cognitive control over eating behaviours, which leaves the individual vulnerable to disinhibited eating when these cognitive processes are disrupted (e.g. as a result of intense emotions) [11].

Persistent distorted thoughts and beliefs, particularly around body dissatisfaction even after weight gain, raises questions about the role of cognitive processes in the development and maintenance of eating disorders [12]. The main cognitive processes that have been heavily researched are executive functioning(e.g. [13]). Executive functions are habitually used as an umbrella term to describe a family of sub-functions that regulate and drive goal-directed and purposeful behaviours, which are crucial for successful day-to-day functioning [14,15]. Research on executive functions and eating disorders has included; attention [16], cognitive flexibility [17], inhibition [18] and working memory [19], resulting in a general consensus that there are executive function deficits in individuals with eating disorders. According to the Continuum Hypothesis [20], individuals with either excessive food intake or food restriction show a significant dysfunctional executive profile [21].

Emphasis has been placed on response inhibition, the ability to suppress inappropriate and unwanted actions. This emphasis is not only as a result of its importance for control of human behaviour, but also because deficient response inhibition has been hypothesized to significantly contribute to several neuropsychiatric disorders [22]. Regarding response inhibition in adults with BN, [23] noted that those with BN responded more impulsively, making more errors on paradigms requiring response inhibition and severity of the condition exaggerated the error rate. Such neuropsychological and imaging work also highlights failures in key frontostriatal brain networks being responsible for impaired behavioural response and the loss of control in eating behaviour in BN. Similarly, decreased activity has been reported in limbic, anterior cingulate cortex and prefrontal regions during target detection task in those with AN [24]. This lack of activation indicates a possible interaction between limbic and frontal networks to exert inhibitory control. The use of less ecologically valid stimuli has determined the importance of the frontal regions. Studies that have implemented such symptom provoking stimuli have noted an increase in frontal brain region activityduring response inhibition tasks (e.g. [25,26]).

Minimal neuropsychological and imaging research has been conducted into executive function and related working memory ability in eating disorders. Due to the complex nature of working memory and its processes [27,28], brain activations during memory tasks are widespread and are dependent upon the task [29]. Research using an n-back working memory task and fMRI noted AN individuals presenting higher activations in temporal and parietal areas especially in the superior temporal gyrus during performance of working memory tasks in comparison with controls [30]. This suggests individuals with AN make additional efforts to achieve normal cognitive performance. This difference subsided after AN weight recovery. However, in an arguable inferior research design, working memory updating was assessed in an undergraduate student population whom scored high and low on the Eating Disorder Examination Questionnaire [31]. Researchers noted that participants did not differ in memory updating abilities regardless of their eating disorder score [32].

One major issue with research in this area is the methodology used to observe the effects of tasks on brain activations. Most, neuropsychological studies on the effect of eating disorders and executive function and working memory have used fMRI. Here we use the temporal precision of event-related potential methodology (ERPs) to precisely track the executive-working memory ability compromised in eating disorders. This will be achieved by employing a three-stimulus Oddball task [33]. During the task, participants are required to discriminate between infrequent target stimuli and frequent standard stimuli. The P3b working memory updating component is elicited during this discrimination. A distracter stimulus is also presented at regular intervals which automatically captures the participants' attention and elicits the P3a ERP component related to attention, novelty detection and response inhibition. Researchers propose that participants who indicate high risk to an eating disorder (based on scores on EDI-3) will have differential activations of ERP brain waves known to be reliable markers of response inhibition (P3a) than those at a low risk. Due to the less reliable and mixed pattern of findings regarding working memory it is also proposed that there will be little or no difference between groups regarding ERPs associated with working memory (P3b). Importantly, due to the importance of the body dissatisfaction in eating disorders and the proposed link to inhibition and impulsivity we predict thought scores on the Body Dissatisfaction scale of the EDI-3 will be associated the P3a ERP component.

2. Method

2.1. Participants

Twelveundergraduate students in Newcastle Upon Tyne were recruited via an opportunity sample. Ages ranged from 18-30 years (mean age=22.42, SD=2.61). Initially, participants were separated into two groups, high predisposition and low predisposition based on their overall score from the questionnaire. As a result there were 5 participants in the high group and 7 in the low group.

2.2. Materials

2.2.1. Predisposition to an Eating Disorder

The Eating Disorder Inventory - 3 [2] was administered to ascertain participants' predisposition to an eating disorder. An overall score of 100 or above was classified as a high predisposition and a score below as a low predisposition. Scores were also obtained for the 12 primary scales: Drive for thinness, Bulimia, Body dissatisfaction, Low self-esteem, Personal alienation, Introceptive deficits, Emotional dysregulation, Perfectionism, Asceticism and Maturity fears. The EDI-3 is one of the most used measures for assessing an eating disorder and has been shown to yield good internal consistency and validity. Internal consistency was found to be between 0.84 and 0.92 for each scale [2].

2.2.2. Working Memory Task

A computerised working memory task, programmed in Eprime was presented. This was a 3 stimulus Oddball task [33] with 4 iterations (approximately 3 minutes each) with a self-paced rest break in between. Participants were required to discriminate between infrequent target stimuli (red circle) and frequent standard stimuli (green square), evoking a P3b component. An infrequent novel distracter stimulus (blue square) was presented, eliciting P3a ERP component relating to attention, response inhibition and novelty detection. On screen instructions were provided at the beginning and during the task to aid completion. The reliability of the Oddball task is great, test-retest correlation coefficients for the Oddball task and P300 amplitudes range from 0.50 to 0.80 [34].

2.2.3. EEG Acquisition

EEG was recorded with a 32 channel electrode cap (BioSemi Active Two) based on an extended 10–20system. The montage comprised four midline sites (Fz, Cz, Pz,Oz), 14 sites over the left hemisphere (Fp1, AF3, F3, F7,FC1, FC5, C3, T7, CP1, CP5, P3, P7, PO3, O1) and 14sites over the right hemisphere (Fp2, AF4, F4, F8, FC2,FC6, C4, T8, CP2, CP6, P4, P8, PO4, O2). The EEG signalwas average electrode referenced, band-pass-filtered at 0.46–30 Hz and digitised at a rate of2,048 per second. Vertical electro-oculogram was recorded for later ocular artifact extraction. EEG epochs recorded from -200 to800. Offline processing of the data was carried out using Edit 4.5 software (Neuroscan).

2.3. Procedure

Participants attended one testing session at a pre-arranged time. Participants were then given the EDI-3 to assess predisposition to an eating disorder. EEG was then set up with the electrode cap and electrodes (including those around the eye) were fitted, the 3 stimulus Oddball task was administered. The task lasted approximately 15 minutes depending upon the participants' self-paced rest breaks.

Finally, participants were given a debrief sheet concluding their part in the study. Results from the questionnaire, the oddball task behavioural data and ERP sextracted from the sites of interest (PZ, CZ and FZ) were entered into SPSS for analyses.

3. Results

Data from EDI-3 and the electrode amplitudes PZ, CZ and FZ were examined using 2 ANOVAs in order to determine the effect of predisposition to an eating disorder on response inhibition and working memory.

A 2 (CZ and FZ electrodes) x 2 (low and high groups) ANOVA yielded no significant results, where the predisposition group did not have a significant effect on the P3a electrodes. In other words, an overall vulnerability to an eating disorder did not significantly affect response inhibition.

Additionally, a 2x2 ANOVA considering PZ and CZ electrodes alongside the low and high groups ascertained that the predisposition group did not significantly affect the P3b electrodes either. Suggesting an overall vulnerability to an eating disorder does not significantly affect working memory.

Both groups were matched on behavioural data from the Oddball task. To verify this, independent samples t-tests, were conducted and concluded that for accuracy, there was no significant difference between low group (mean = 0.98, SD = 0.02) and high group (mean = 0.98, SD = 0.02). For reaction times, there was also no significant difference between low group (mean = 412.06, SD = 26.58) and high group (mean = 421.09, SD = 60.75).

Further subsidiary analysis was undertaken to correlate electrodes and the 12 scales on the EDI-3. Particular interest was of the Body Dissatisfaction scale. All variables were non-significant with the exception of the body dissatisfaction scale and the P3a FZ electrode which was positively correlated (see Fig 1). r (10) = .596, p = .041, d = 1.484. The greater the body dissatisfaction score, the greater the amplitude in the frontal area of the brain during response inhibition task. This result attributed to a large effect size, d = 1.484, suggesting that the significant difference is in fact large, making the result more relevant.

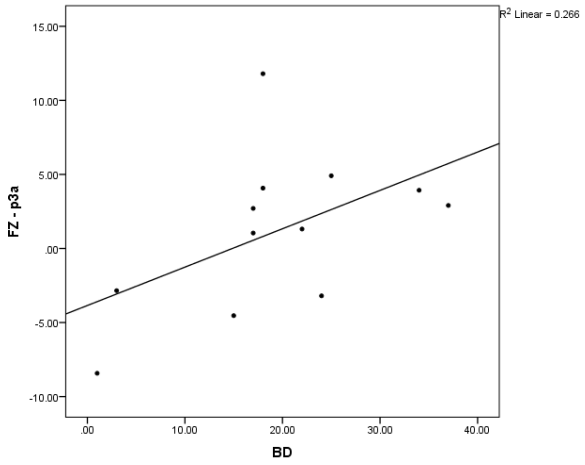

Figure 1. Positive correlation between FZ electrode and Body Dissatisfaction score

Head maps were created based on a median split of body dissatisfaction scores to demonstrate activations during P3a and P3b aspects of the oddball task (see Fig 2.).

Both images demonstrate great activation in the central brain area but the higher body dissatisfaction group shows greater activation towards the frontal region than the lower group during the executive component of the task. There was a significant effect based on correlational analysis suggesting individuals with a high body dissatisfaction score have greater hyperactivity in the frontal area of their brain (an executive control region) during the response inhibition section of the Oddball task.

Although correlational analysis rendered no significant results for the memory component, Figure 2 suggests greater variability in activated brain areas for the high body dissatisfaction group. There is a wider spread of activation than the low group, with more activation towards the frontal brain areas. In addition, there is less parietal activation than in both the low group and control (all participants); however, activation is less overall for the high group. Such imaging suggests that individual's with a high body dissatisfaction score require more area activations in the brain during the completion of a

memory component than those with a low score. Additionally, those with a high score appear to rely more on fronto-central areas of the brain

4. Discussion

The current study aimed to investigate the effect of an overall predisposition to an eating disorder alongside individual risk factors on known ERP measures of response inhibition (P3a) and working memory (P3b). Based on overall scores of the EDI-3 the results provided no evidence for an interaction between an overall predisposition to an eating disorder and P3a and P3b ERP amplitudes. It was hypothesised that there would be an effect on P3a electrodes (response inhibition) and little/no effect on the P3b (working memory). Arguably using a crude measure of total score on the EDI-3 conceals the underlying factors responsible for individual difference in brain markers of inhibition and working memory. Indeed, the current study as predicted did ascertain a significant positive correlation between Body Dissatisfaction scores and the P3a FZ electrode amplitudes (location where this component is centered). Individuals with a high score on body dissatisfaction scale showed greater activation towards the frontal region than those with a low score during the executive function task, i.e. the greater the score the greater hyperactivity in the frontal area of the brain during response inhibition task.

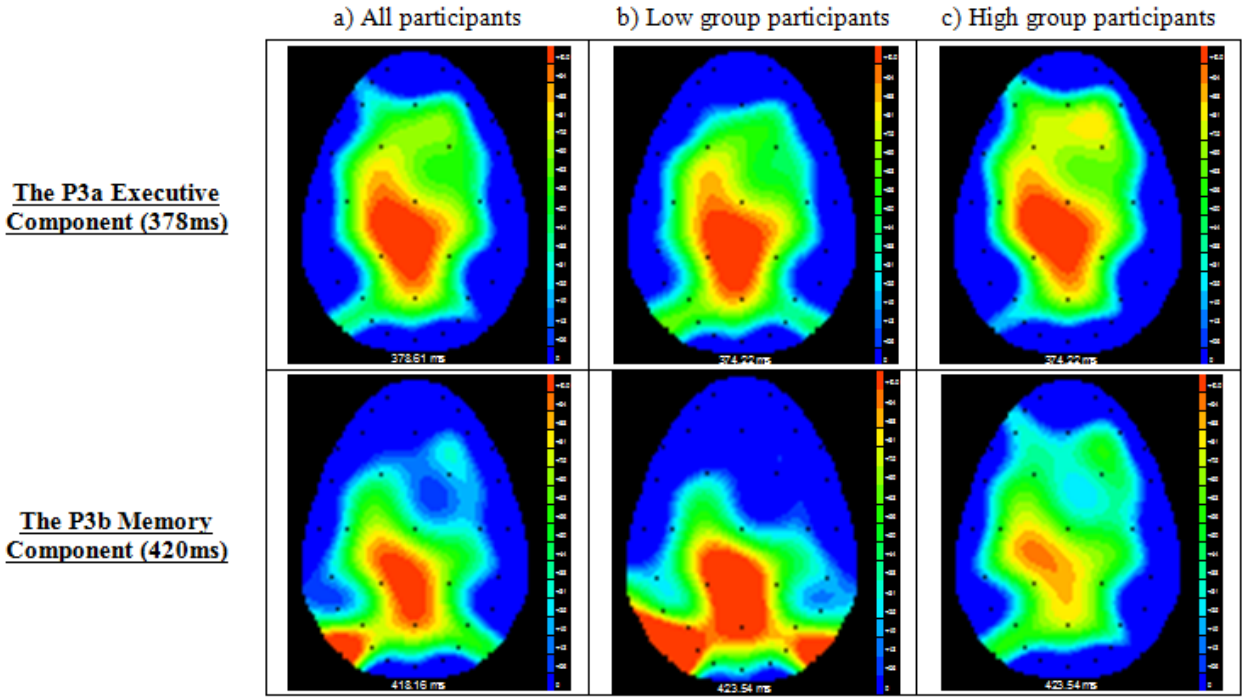

Figure 2. Average ERPs for the executive P3a component and working memory P3b component. a) All participants, b) low group and c) high groupbased on median split of body dissatisfaction data. Note analysis is based on continuous data i.e. correlations between raw BD scores and ERPs

No significant effects were found in the working memory component. As a result of the significant positive correlation of body dissatisfaction scores and response inhibition, head maps were created. Head maps for the working memory component were included to see if body dissatisfaction as a predisposition had any effect on the distribution of ERPs. From this it can be seen that there is a wider spread of activation for the high body dissatisfaction group, rather than concentrated activations in the parietal region for working memory. The possible implications of this will be discussed further. The current study's results are both consistent and inconsistent with present literature in this area.

Despite this research being novel in its field, there are some links to earlier behavioural and neuroscience work in the area. Body dissatisfaction theoretically promotes unhealthy dieting behaviours such as restriction or binging due to violations of strict dietary rules. This shifts reliance on physiological to cognitive control over eating behaviours and consequently leaves a vulnerability to disinhibted eating [11]. It is this lack of inhibitory control that is linked to eating disorders and executive function deficits. A task requiring inhibitory control showed an increase in activations in the dorsolateral prefrontal cortex, an executive function region, for those with an eating disorder [35]. This suggested inefficient or possibly compensatory brain activations (i.e. recruitment of additional brain regions) [36]. This research supports results found in the current study where, for high scores on body dissatisfaction there was greater activation/hyperactivity towards the frontal region (specifically around the frontal FZ electrode) during the response inhibition aspect of the task than for low scores. Such hyper-frontal activity is indicative ofan inability to ignore task irrelevant stimuli present during the task [see [37,38] for this measure used in other psychological domains].

Another possible explanation for positive correlation between the P3a component and Body Dissatisfaction score is the role of Dopamine. P3a activations have been associated with frontal lobe related attention mediated by dopaminergic activity [39], where increased activations are often as a result of greater Dopamine levels. Reference [40] looked into P3a amplitudes of healthy controls, individuals with restless leg syndrome and Parkinson's

disease. Restless leg syndrome is thought to originate from dopaminergic deficits; these deficits are greater in Parkinson's disease. It was found that P3a amplitudes were reduced for restless leg syndrome and virtually eliminated for Parkinson's disease; the assumption was that a lack of Dopamine was the cause. Additionally it has been found that individuals with Parkinson's disease indicate impairments in inhibitory control [41]. Together such research argues for the use of the P3a as a marker of dopamine function.

The current study looked at memory updating in relation to body dissatisfaction. Those who scored high had reduced parietal amplitudes and increased frontocentral activations in comparison to controls during the task. It has been concluded that high body dissatisfaction elicits compensatory brain activations during memory updating. Dopamine dysfunction in the frontostriatal systems may promote impulsivity by impairing the flexible memory updating of task relevance and inducing susceptibility to distract in working memory [42]. This suggests that the current study's findings are a result of Dopamine activity which promotes frontal activations. This accounts for the widespread activation and to compensate for this, the parietal region has less activation during working memory for those with high body dissatisfaction. In support of a Dopamine interaction, dopaminergic medications have been found to improve the updating of information in working memory in patients with ADHD [42]. As a result, future research analysis should include other electrode amplitudes to ascertain if widespread activation is significant in body dissatisfaction groups. The current study did not do this and only considered the PZ and CZ electrodes associated with central parietal regions where the effect is usually centered.

In summary, the current study's findings have both supported and contradicted present research. Importantly, findings have implicated the role of body dissatisfaction on modulating the P3a and P3b amplitudes. It was noted that the greater the body dissatisfaction score, the greater the P3a amplitude in the frontal regions during response inhibition. Consequently, the conclusion was of this being a hyperactivation, suggesting over compensation for response inhibition deficits. A large effect size was also noted (d = 1.484) despite only having a small sample of 12 participants. Additionally, although not significant, body dissatisfaction scores were interpreted to have an effect on working memory component with broad activations across from parietal to frontal regions. If body dissatisfaction can be identified as an indicator of greater response inhibition and differential working memory brain activations, it could consequently be implicated in early screening programmes to identify individuals with a potential eating disorder. The novel aspect of this study has helped to broaden potential future research and possible treatments. It is important to try and consider the effect of predisposing factors of an eating disorder on executive functions, not just the eating disorder itself.

References

[1] Bemporad, J.R, "Cultural and historical aspects of eating disorders," *Theoretical Medicine*, *18*(4). 401-420. 1997.

[2] Garner, D.M, EDI 3: Eating Disorder Inventory-3: Professional Manual, Psychological Assessment Resources. 2004.

[3] American Psychiatric Association, *Diagnostic and statistical manual of mental disorders*(5th ed.), American Psychiatric Publishing, Arlington, VA. 2013.

[4] Corstorphine, E, "Cognitive–emotional–behavioural therapy for the eating disorders: Working with beliefs about emotions," *European Eating Disorders Review*, *14*(6). 448-461. Oct.2006

[5] McKnight Investigators, "Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study," *American Journal of Psychiatry*, *160*(2). 248-254. Feb.2003.

[6] Obeid, N., Buchholz, A., Boerner, K.E., Henderson, K.A., and Norris, M, "Self-esteem and social anxiety in an adolescent female eating disorder population: age and diagnostic effects," *Eating disorders*,*21*(2). 140-153. Feb.2013

[7] Stice, E., Davis, K., Miller, N.P., and Marti, C.N, "Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study," *Journal of abnormal psychology*, *117*(4). 941. Nov.2008.

[8] Fairburn, C.G., Cooper, Z., Doll, H.A., and Davies, B.A, "Identifying dieters who will develop an eating disorder: a prospective, population-based study," American Journal of Psychiatry. Dec.2014.

[9] Shafran, R., Cooper, Z. and Fairburn, C.G, "Clinical perfectionism" is not "multidimensional perfectionism: a reply to Hewitt, Flett, Besser, Sherry and McGee," Behaviour Research and Therapy, 41. 1217-1220. 2003

[10] Stice, E, "A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect," *Journal of abnormal psychology*, *110*(1). 124. Feb.2001.

[11] Stice, E., and Shaw, H.E, "Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings," *Journal of psychosomatic research*, *53*(5). 985-993. Nov.2002.

[12] Garner, D.M., and Bemis, K.M, "A cognitive-behavioral approach to anorexia nervosa," *Cognitive therapy and research*, *6*(2). 123-150. Jun.1982.

[13] Kothari, R., Solmi, F., Treasure, J., and Micali, N, "The neuropsychological profile of children at high risk of developing an eating disorder," *Psychological medicine*, *43*(07). 1543-1554. Jul.2013.

[14] Parrish, J., Geary, E., Jones, J., Seth, R., Hermann, B., and Seidenberg, M, "Executive functioning in childhood epilepsy: parent-report and cognitive assessment," *Developmental Medicine & Child Neurology*,*49*(6). 412-416. Jun.2007.

[15] Riccio, C.A., and Gomes, H, "Interventions for executive function deficits in children and adolescents," *Applied Neuropsychology: Child*,*2*(2). 133-140. Jan.2013.

[16] Dobson, K.S., and Dozois, D.J, "Attentional biases in eating disorders: A meta-analytic review of Stroop performance," *Clinical Psychology Review*, *23*(8). 1001-1022. Jan.2004.

[17] Tchanturia, K., Davies, H., Roberts, M., Harrison, A., Nakazato, M., Schmidt, U., Treasure, J., and Morris, R, "Poor cognitive flexibility in eating disorders: examining the evidence using the Wisconsin Card Sorting Task," *PLoS One*, *7*(1). Jan.2012.

[18] Galimberti, E., Martoni, R.M., Cavallini, M.C., Erzegovesi, S., and Bellodi, L, "Motor inhibition and cognitive flexibility in eating disorder subtypes," *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *36*(2). 307-312. Mar.2012.

[19] Kemps, E., Tiggemann, M., Wade, T., Ben-Tovim, D., and Breyer, R, "Selective working memory deficits in anorexia nervosa," *European Eating Disorders Review*,*14*(2). 97-103. Mar.2006.

[20] Scarano, G.M., and Kalodner-Martin, C.R, "A description of the continuum of eating disorders: Implications for intervention and research," *Journal of Counseling & Development*,*72*(4). 356-361. Apr.1994.

[21] Williamson, D.A., Womble, L.G., Smeets, M.A., Netemeyer, R.G., Thaw, J.M., Kutlesic, V., and Gleaves, D.H, "Latent structure of eating disorder symptoms: A factor analytic and taxometric investigation," *American Journal of Psychiatry*, *159*(3). 412-418. Mar.2002.

[22] Simmonds, D.J., Pekar, J.J., and Mostofsky, S.H, "Meta-analysis of Go/No-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent," *Neuropsychologia*, *46*(1). 224-232. Jan.2008.

[23] Marsh, R., Maia, T.V., and Peterson, B.S, "Functional disturbances within frontostriatal circuits across multiple childhood psychopathologies," *The American journal of psychiatry*, *166*(6). 664-674. Jun.2009.

[24] Zastrow, A., Kaiser, S., Stippich, C., Walther, S., Herzog, W., Tchanturia, K., and Friederich, H.C, "Neural correlates of impaired cognitive-behavioral flexibility in anorexia nervosa," American Journal of Psychiatry, 166(5). 608-616. May.2009.

[25] Baddeley, A.D., and Hitch, G, "Working memory," *Psychology of learning and motivation*, *8*. 47-89. 1974.

[26] Baddeley, A, "The episodic buffer: a new component of working memory?" *Trends in cognitive sciences*, *4*(11). 417-423. Oct.2000.

[27] Brooks, S.J., Owen, G.O., Uher, R., Friederich, H.C., Giampietro, V., Brammer, M., and Campbell, I.C, "Differential neural responses to food images in women with bulimia versus anorexia nervosa," *PLoS One*,*6*(7). Jul.2010.

[28] Kullmann, S., Giel, K.E., Hu, X., Bischoff, S.C., Teufel, M., Thiel, A., and Preissl, H, "Impaired inhibitory control in anorexia nervosa elicited by physical activity stimuli," *Social cognitive and affective neuroscience*, *9*(7). 917-923. Jan.2014.

[29] Pessoa, L., Gutierrez, E., Bandettini, P.A., and Ungerleider, L.G, "Neural correlates of visual working memory: fMRI amplitude predicts task performance," *Neuron*,*35*(5). 975-987. Aug.2002.

[30] Castro-Fornieles, J., Caldú, X., Andrés-Perpiñá, S., Lázaro, L., Bargalló, N., Falcón, C., and Junqué, C, "A cross-sectional and follow-up functional MRI study with a working memory task in adolescent anorexia nervosa," *Neuropsychologia*,*48*(14). 4111-4116. Dec.2010.

[31] Fairburn, C.G., and Beglin, S.J, "Assessment of eating disorders: Interview or self-report questionnaire?," *International Journal of Eating Disorders*, *16*(4). 363-370. Dec.1994.

[32] Fenton, O., and Ecker, U.K, "Memory updating in sub-clinical eating disorder: Differential effects with food and body shape words," *Eating behaviors*, *17*. 103-106. Apr.2015.

[33] Polich, J, *Theoretical overview of P3a and P3b*, Springer, US, 2003, 83-98.

[34] Walhovd, K.B., and Fjell, A M, "One-year test–retest reliability of auditory ERPs in young and old adults," *International Journal of Psychophysiology*,*46*(1). 29-40. Oct.2002.

[35] Lock, J., Garrett, A., Beenhakker, J., and Reiss, A.L, "Aberrant brain activation during a response inhibition task in adolescent eating disorder subtypes," American Journal of Psychiatry. Jan.2011.

[36] Han, S.D., Bangen, K.J., and Bondi, M.W, "Functional magnetic resonance imaging of compensatory neural recruitment in aging and risk for Alzheimer's disease: review and recommendations," *Dementia and geriatric cognitive disorders*,*27*(1). 1. Feb.2009.

[37] Barron, E., Riby, L.M., Greer, J., and Smallwood, J, "Absorbed in thought the effect of mind wandering on the processing of relevant and irrelevant events," *Psychological science*. Mar.2011.

[38] Riby, L M, "The Joys of Spring: Changes in Mental Alertness and Brain Function," *Experimental psychology*,*60*(2). 71-79. Jan.2013.

[39] Polich, J., and Criado, J.R, "Neuropsychology and neuropharmacology of P3a and P3b," *International Journal of Psychophysiology*, *60*(2). 172-185. May.2006.

[40] Trenkwalder, C., and Winkelmann, J, "Pathophysiology of the restless legs syndrome," *Sleep and movement disorders, Philadelphia: Butterworth/Henemann*. 322-32. 2003.

[41] Bokura, H., Yamaguchi, S., and Kobayashi, S, "Event-related potentials for response inhibition in Parkinson's disease," *Neuropsychologia*,*43*(6). 967-975. 2005.

[42] Frank, M.J., Moustafa, A.A., Haughey, H.M., Curran, T., and Hutchison, K.E, "Genetic triple dissociation reveals multiple roles for dopamine in reinforcement learning," *Proceedings of the National Academy of Sciences*, *104*(41). 16311-16316. Aug.2007.